CHAPTER 14 Gastrointestinal (GI) Pathology

1 Gastrointestinal (GI) Pathology Overview

- The gastrointestinal system consists of:
  - The GI tract—oral cavity, esophagus, stomach, small intestine, colon, and anal canal (covered in order).
  - Accessory organs—liver, pancreas, and biliary tract.
- GI tract diseases fall into several categories:
  - **Inflammatory**
    Esophagitis
  - **Vascular**
    —Mallory-Weiss syndrome
    —Esophageal varices
  - **Neoplastic**
  - Squamous cell carcinoma
  - Adenocarcinoma

2 Oral Cavity and Salivary Gland Disorders

2.1 Cleft Lip and Palate

Most common congenital disorder of oral cavity:

- About 1:800 live births:
  - Cleft lip and palate (50%)
  - Cleft lip alone (25%; male > female)
  - Cleft palate alone (25%)
- Genetic factors involved: Presence in subsequent siblings (3%)
- More common in the Caucasian population
- Complications:
  - Malocclusion
  - Eustachian tube dysfunction (chronic *otitis* media)
  - Speech problems
- Pathogenesis: Failure of fusion of facial processes
- Treatment: Surgery

USMLE® Key Concepts

For Step 1, you must be able to:

- ▶ Identify various oropharyngeal pathologies.
- ▶ Differentiate among various esophageal pathologies.
- ▶ Explain the pathogenesis of various gastric pathologies.
- ▶ Identify various mechanical, malabsorptive, and neoplastic processes of the small and large intestine.
- ▶ Differentiate among different types of malabsorbtive and diarrheal conditions, and identify the causative organism responsible for these pathological conditions.

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2.2 Common Infections of the Oral Cavity

▼ Table 14-2.2 Infections of Oral Cavity

INFECTION	PATHOGEN	FEATURES
Viral		
Exudative tonsillitis	Viruses (most cases)	Culture is necessary to differentiate bacterial versus viral infection
Hairy leukoplakia		• Glossitis associated with bilateral white excrescences on lateral border of tongue • Pre-AIDS-defining lesion
Herpes labialis	HSV type 1	• Recurrent vesicular lesions on the lips (virus remains dormant in cranial sensory ganglia) • Reactivated by stress, sunlight, and menses • Treatment: Oral acyclovir, valacyclovir, famciclovir; topical acyclovir, penciclovir
Mumps	Paramyxovirus	• Bilateral parotitis (70%) with increased serum amylase • Complications: Meningoencephalitis, unilateral orchitis or oophoritis, pancreatitis
Herpangina	Coxsackievirus	• Occurs in children • Typically occurs in epidemics during the summer • Painful vesicles or small white papules occur on an erythematous base typically at the junction of the soft and hard palate
Hand-foot-and-mouth disease	Coxsackievirus	• Occurs in young children • Vesicles located in mouth and distal extremities
Bacterial		
Cervicofacial actinomycosis	*Actinomyces israelii*	• Draining sinus tract from facial or cervical area • "Sulfur granules" in pus contain gram-positive, branching filamentous anaerobic bacteria • Often follows extraction of an abscessed tooth • Treatment: ampicillin, penicillin G
Diphtheria	*Corynebacterium diphtheriae*	• Toxin produces "shaggy" gray pseudomembrane in posterior pharynx and upper airways • Treatment: Erythromycin
Peritonsillar abscess	*Streptococcus pyogenes*	• Uvula deviates to contralateral side; "hot potato" voice; foul-smelling breath • Complication due to tonsillitis • Treatment: Surgical drainage of pus; penicillin G or V; add clindamycin for serious invasive infections
Ludwig angina	Aerobic/anaerobic *Streptococcus, Eikenella corrodens*	• Cellulitis involving the submaxillary and sublingual space; follows fascial planes and may spread into pharynx, carotid sheath, superior mediastinum • Causes: Dental extraction (most common), trauma to floor of mouth • Treatment: Surgical drainage; clindamycin + metronidazole
Pharyngitis	*S. pyogenes*	• Associated with tonsillitis • Potential for acute rheumatic fever and glomerulonephritis • Treatment: Penicillin V
Scarlet fever	*S. pyogenes*	• Pharyngitis, tonsillitis, glossitis • Erythrogenic toxin produces rash on skin and tongue (initially white and then strawberry colored) • Increased risk for glomerulonephritis • Nephritogenic strains pose no risk for acute rheumatic fever • Treatment: Penicillin G or V

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▼Table 14-2.2 Infections of Oral Cavity *continued*

INFECTION	PATHOGEN	FEATURES
Bacterial *continued*		
Sialadenitis	*Staphylococcus aureus*	• Bacterial inflammation of major salivary gland • Secondary to a calculus, which obstructs the duct in postoperative patients • Treatment: Oxacillin, nafcillin if methicillin susceptible; TMP-SMX if community-acquired methicillin resistant; vancomycin if methicillin resistant in hospital
Congenital syphilis	*Treponema pallidum* (spirochete)	• Abnormalities involving incisors (notched and tapered like a peg) and molar teeth (resemble mulberries) • Treatment: Aqueous crystalline penicillin G
Acute necrotizing gingivitis (Vincent angina)	Anaerobes: *Prevotella, Fusobacterium; spirochetes*	• Anaerobic bacterial infection of gingiva • Necrosis of interdental papilla with punched out lesions covered by a grayish pseudomembrane • Treatment: Penicillin or metronidazole
Fungal		
Oral thrush	*Candida albicans* (yeast)	• May occur in neonates, immunocompromised patients (common pre-AIDS-defining lesion), diabetes mellitus, and following antibiotic therapy • Treatment: Fluconazole, itraconazole

2.3 HIV-Related Oral Infections

- Candidiasis (most common)
- Aphthous ulcers (stomatitis; canker sores):
  - Unknown origin:
    —Virus versus immunologic
    —Often stress-induced
  - Painful ulcers covered by a shaggy gray exudate
- Hairy leukoplakia (glossitis due to Epstein-Barr virus)
- Kaposi sarcoma:
  - Hard palate is the most common location
  - Due to human herpesvirus 8 (HHV8)
- Dental caries:
  - *Streptococcus mutans* produces acid from sucrose fermentation, which erodes enamel and exposes underlying dentine
  - Fluoride prevents dental caries—excess fluoride causes chalky discoloration of the teeth

Important Concept

Pre-AIDS-defining lesions:

- Thrush
- Hairy leukoplakia
- Aphthous ulcers

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2.4 Noninfectious Ulceration in the Oral Cavity

- Pemphigus vulgaris and mucous membrane pemphigoid (both are immunologic skin disorders)
- Erythema multiforme:
  - Hypersensitivity reaction against *Mycoplasma* or drugs such as sulfonamides
  - Called Stevens-Johnson syndrome when it involves the mouth
- Aphthous ulcers (stomatitis)
- Behçet syndrome
  - Epidemiology (combination of environmental + genetic factors):
    —Human leukocyte antigen (HLA)-B51, HLA-B27
    —May be precipitated by herpes simplex or parvovirus
    —High incidence of Turkish and Eastern Mediterranean descent
  - Pathophysiology: Immune complex small vessel vaculitis
  - Clinical findings:
    —Recurrent aphthous ulcers, genital ulcerations
    —Uveitis, erythema nodosum
    —Attacks last one to four weeks
  - Treatment:
    —Anti-inflammatory medications
    —Colchicine, thalidomide

2.5 Pigmentation Abnormalities of the Mouth and Oral Cavity

- Peutz-Jeghers syndrome: Melanin pigmentation of the lips and oralmucosa
- Addison disease: Feedback mechanism in which there is an increase in adrenocorticotropic hormone release from proopiomelanocortin resulting in melanin pigmentation on the buccal mucosa
- Lead poisoning: Lead deposits along the gingival margins in adults with gingivitis

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▲ **Figure 14–2.5** Peutz-Jeghers Syndrome

2.6 Tooth Discoloration

- **Tetracycline:**
  - Drug discolors newly formed teeth
  - Not recommended in a child <12 years of age
- **Excess fluoride:**
  - Fluoride is used in toothpaste to prevent dental caries
  - Excess may cause teeth to be mottled, chalky white discoloration
- **Congenital erythropoietic porphyria:**
  - Porphyrin deposits in the teeth
  - Reddish-brown discoloration

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2.7 Pathological Findings of the Tongue

- **Macroglossia:**
  - Myxedema deposit as a result of hypothyroidism
  - Down syndrome
  - Acromegaly
  - Systemic amyloidosis
  - Mucosal neuromas in multiple endocrine neoplasia type IIb
- **Glossitis:**
  - Definition: Sore, beefy red tongue with or without papillary atrophy
  - Causes:
    - Long-standing iron deficiency as seen with Plummer-Vinson syndrome
    - Vitamin B12 or folate deficiencies known to cause megaloblastic anemias
    - Vitamin C deficiency known to cause scurvy
    - Vitamin B2 (riboflavin) deficiency
    - Vitamin B3 (niacin) deficiency known to cause pellagra
    - Hairy leukoplakia as a result of Epstein-Barr virus infection
- **Leukoplakia and erythroplakia:**
  - Definitions:
    - Leukoplakia: "White patch"
    - Erythroplakia: "Red patch"
    - Leukoerythroplakia: Combination of the two
  - Lesions initially show squamous hyperplasia of the epidermis:
    - May progress into squamous dysplasia or invasive squamous cell carcinoma (SCC)
    - Erythroplakia has ~60% rate of progression to oral cancer
    - Leukoplakia has ~30% rate of progression to oral cancer
  - Locations:
    - Vermilion border of lower lip is the most common site
    - Buccal mucosa, hard and soft palates, and the floor of the mouth
  - Etiology:
    - Chronic irritation from dentures as an example
    - All forms of tobacco use
    - Alcohol abuse
    - Human Papillomavirus (HPV)
  - Always biopsy these lesions because of the high risk for progression to oral cancer
- **Lichen planus:**
  - Often associated with Wickham striae (fine, white, lacy lesions) on the buccal mucosa
  - May be associated with squamous cell carcinoma

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2.8 Salivary Gland Pathology

- **Sjögren syndrome:**
  - Epidemiology and pathogenesis:
    - Female dominant autoimmune disease associated with rheumatoid arthritis
    - Autoimmune destruction of minor salivary glands and lacrimal glands
  - Clinical findings:
    - Xerostomia:
      - Dry mouth may result in infection of the oral cavity such as dental caries
      - Patient may complain of difficulty with consuming dry foods such as crackers
    - Keratoconjuctivitis sicca
      - Dry eyes described as "sand in my eyes"
      - Lack of tears renders the conjunctiva susceptible to injury
  - Laboratory findings:
    - ANA +
    - Serum rheumatoid factor (RF) + in 90% of cases
    - Anti-SS-A (anti-Ro) antibodies + (70%–95% of cases)
    - Anti-SS-B (anti-La) antibodies + (60%–90% of cases)
  - Confirm with a lip biopsy, which must demonstrate lymphoid destruction of the minor salivary glands
  - Treatment:
    - Artificial tears
    - Pilocarpine or cyclosporine eye drops
    - Cevimeline (cholinergic agent with muscarinic agonist activity used for dry mouth)
- **Pleomorphic (mixed) adenoma:**
  - *Most common benign tumor of major and minor salivary glands*
  - Parotid gland is the most common site
  - Female dominant
  - Painless, moveable mass at the angle of the jaw
  - Epithelial cells intermixed with myxomatous and cartilaginous stroma
  - Tumor projections through capsule increase risk of recurrence
  - May transform into a malignant tumor
  - Facial nerve involvement is a sign of malignancy
- **Warthin tumor (papillary cystadenoma lymphomatosum):**
  - Tumor is a *benign parotid gland tumor and tends to be male dominant*
  - Smokers have an increased risk of developing these tumors
  - Heterotopic salivary gland tissue is trapped in a lymph node
  - Cystic glandular structures are located within benign lymphoid tissue
- **Mucoepidermoid carcinoma**
  - *Most common malignant salivary gland tumor*
  - Most commonly located in the parotid gland
  - Mixture of neoplastic squamous and mucus-secreting cells

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2.9 Tumors of the Oral Cavity

- **Benign Tumors**
  - Squamous papillomas
    - Most common benign tumors in oral cavity
    - Exophytic tumor with a fibrovascular core
    - May occur on the tongue, gingiva, palate, or lips
  - Ameloblastoma
    - Arises from enamel organ epithelium or a dentigerous cyst
    - Located in the mandible
      - Produces a radiolucency in bone that has a "soap bubble" appearance
      - Locally invasive but does not metastasize
- **Malignant Tumors**
  - *Majority are well-differentiated squamous cell carcinomas*
  - Incidence in men greater than in women
  - Risk factors:
    - HPV is the most common risk factor
    - Use of tobacco products
    - Synergistic use of alcohol with smoking
    - Chronic irritation from dentures
    - Lichen planus
  - Cancer sites in descending order:
    - Vermilion border of lower lip
    - Floor of mouth
    - Lateral border of tongue
  - Metastasis: Tonsillar node (superior juglar node)
  - *Verrucous carcinoma:* Associated with smokeless tobacco
  - *Basal cell carcinoma:*
    - Most common cancer of the upper lip
    - Associated with exposure to UV-B light rays
  - Treatment:
    - Surgery and radiation and chemotherapy in advanced cases
    - HPV vaccination is protective against developing HPV oropharyngeal cancers

Important Concept

Dentigerous Cyst

- Derives from epithelium elements of dental origin
- Associated with the crown of an unerupted or impacted third molar
- Associated with ameloblastoma in 15%–30% of cases

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6 Diarrhea

▼Table 14–6.0A Types and Characteristics of Diarrhea

Type	Characteristics	Causes	Screening Tests
Invasive	Pathogens invade enterocytes Low-volume diarrhea	*Shigella* spp. *Campylobacter jejuni*	Fecal smear for leukocytes: positive in most cases
	Diarrhea with blood and leukocytes (i.e., dysentery)	*Entamoeba histolytica*	Order stool culture and stool for O&P
Secretory	Loss of isotonic fluid High-volume diarrhea Mechanisms: • Laxatives • Enterotoxins stimulate Cl- channels regulated by cAMP and cGMP • Serotonin increases bowel motility No inflammation in bowel mucosa	Laxatives: Danger of melanosis coli (black bowel syndrome) with use of phenanthracene laxatives Production of enterotoxins: • *Vibrio cholerae* • Enterotoxigenic *E. coli* Increased serotonin: carcinoid syndrome	Fecal smear for leukocytes: negative Increased 5-HIAA: carcinoid syndrome Stool Osmolal Gap (SOG) <50 mOsm/kg SOG = StoolOsm – (2* [Stool NA^+] + (Stool K^+])
Osmotic	Osmotically active substance is drawing hypotonic salt solution out of bowel High-volume diarrhea No inflammation in bowel mucosa	Disaccharidase deficiency Stunned gut in giardiasis Ingestion of poorly absorbable solutes (e.g., magnesium sulfate laxatives)	Fecal smear for leukocytes: negative Stool Osmolal Gap (SOG) >125 mOsm/kg

▼Table 14–6.0B Pathogens and Diarrhea

Pathogen	Discussion
Viruses	
Cytomegalovirus	Common cause of diarrhea in AIDS when CD4 T_H cell count <50–100 cells/mm^3
	Treatment: ganciclovir
Norwalk virus	Most common cause of adult gastroenteritis
	Nausea, vomiting, diarrhea that resolves in 12–24 hours
	Can occasionally be fatal
	Fecal-oral transmission
	Common infection on cruise ships
	Treatment: supportive
Rotavirus	Most common cause of childhood diarrhea; particularly occurs in winter months Fecal-oral transmission
	Damages ion transport pump in small intestine; secretory diarrhea
	Rotazyme test on stool establishes diagnosis
	Rotavirus vaccine highly effective in prevention; oral vaccine
	Treatment: oral hydration; nitazoxanide

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▼Table 14–6.0B Pathogens and Diarrhea *(continued)*

Pathogen	Discussion
Bacteria	
Bacillus cereus	Gram-positive rod
	Food poisoning with preformed toxin
	Associated with reheated fried rice or tacos
	Self-limited
Campylobacter jejuni	Curved or S-shaped gram-negative rod
	Animal reservoirs: cattle, chicken, puppies (common source for children)
	Fecal-oral transmission via contaminated water, poultry, or unpasteurized milk
	Most common food-borne illness and invasive enterocolitis in U.S.
	Invasive and secretory enterocolitis: dysentery (bloody diarrhea) with crypt abscesses and ulcers resembling ulcerative colitis; high fever and cramping abdominal pain; organisms in stool with blood and leukocytes
	Complications: Guillain-Barré syndrome (antibodies cross-react with neurons); hemolytic uremic syndrome; HLA-B27 positive seronegative spondyloarthropathy
	Treatment: azithromycin
Clostridium botulinum	Gram-positive rod
	Adult food poisoning with preformed toxin (blocks release of acetylcholine release in presynaptic terminal of neuromuscular junction in autonomic nervous system; causes descending paralysis, mydriasis, dry mouth
	Treatment: trivalent antitoxin
	Infant food poisoning often contracted by eating spores in honey (lack protective bacteria); floppy baby with constipation
Clostridium difficile	Gram-positive rod Associated with pseudomembranous colitis; the most common cause of nosocomial diarrhea; secretory type of diarrhea
	Normally present in 3% of people; carrier rate increases to >20% in hospitalized patients (related to contact with spores in environment and fecal-oral contamination)
	Antibiotic-induced in 65%-90% of cases; antibiotics (e.g., ampicillin, quinolones, clindamycin) cause overgrowth of toxin-producing *C. difficile* in colon; toxins A and B release proinflammatory mediators and cytokines that attract neutrophils and stimulate excess fluid secretion (watery diarrhea)
	Pseudomembrane covers colon mucosa; composed of cellular debris, leukocytes, fibrin, and mucin
	Person-to-person induced in 30% of cases
	Nonspecific lab findings: neutrophilic leukocytosis with left shift; fecal leukocytes; and decreased serum albumin
	• Cytotoxin assay of diarrheal (not solid) stool has greater specificity of 75%–100% when compared to culture of stool, which is 75%–80% for securing the diagnosis • Glutamate dehydrogenase antigen test is also used and has excellent sensitivity and specificity enzyme present in strains of *C. difficile*)
	• *Treatment:* Metronidazole; vancomycin produces resistant strains • Increased mortality with increased age and increased virulence of antibiotic-resistant strains

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▼Table 14–6.0B Pathogens and Diarrhea *(continued)*

Pathogen	Discussion
Bacteria ***(continued)***	
Escherichia coli	Gram-negative rod
	Enterotoxigenic *E. Coli* (ETEC) causes traveler's diarrhea with the presence of two toxins that increase the conductance of chloride into the luminal gut (secretory type of diarrhea): • Heat Labile Toxin (LT) which works through adenylate cyclase • Heat Stable Toxin (ST) which works through guanylate cyclase
	Treatment: ciprofloxacin
	STEC (O157:H7 serotype): contracted by eating undercooked beef
	• Shiga-toxin producing *E. coli* (STEC) (O157:H7) contracted by consuming undercooked beef • Responsible for large outbreaks of bloody diarrhea and hemolytic uremic syndrome (HUS)
	Antibiotics *not* recommended; may enhance toxin release
Mycobacterium avium-intracellulare complex (MAC)	Acid-fast rods Causes diarrhea with malabsorption in AIDS (CD4 count <50 cells/mm^3)
	Foamy macrophages in lamina propria simulate Whipple disease
Mycobacterium tuberculosis	Acid-fast organisms swallowed from primary focus in lung
	Invade Peyer patches
	Circumferential spread in lymphatics leads to stricture formation
Salmonella species	Gram-negative rod
	Pathogenic *Salmonella: S. typhi, S. paratyphi, S. enteritidis*
	Animal reservoirs: turtles, hamsters, lizards
	Salmonella enteritidis enterocolitis: Second most common food-borne illness in U.S.; contracted by eating raw or undercooked egg products and poultry, raw milk and milk products, or drinking contaminated water
	Treatment: ciprofloxacin or levofloxacin
	Typhoid fever caused by *S. typhi*: Week 1: invades Peyer patches and produces sepsis (blood culture best for diagnosis) Week 2: diarrhea (positive stool culture); classic triad of bradycardia, neutropenia, splenomegaly
	Treatment: treat if symptomatic with fluoroquinolone; antibiotics do *not* shorten the illness and may increase frequency of carrier states
	Chronic carrier state due to gallbladder disease: cholecystectomy
Shigella dysenteriae and Shigella sonnei	Gram-negative rod
	No animal reservoirs
	Highly infectious; children in day care centers; mental institutions
	Mucosal ulceration, pseudomembranous inflammation in rectosigmoid, dysentery
	Association with HLA-B27 positive seronegative spondyloarthropathy
	Treatment: treat if symptomatic with fluoroquinolone or azithromycin

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▼Table 14–6.0B Pathogens and Diarrhea *(continued)*

Pathogen	Discussion
Bacteria ***(continued)***	
Staphylococcus aureus	Gram-positive coccus
	Food poisoning with preformed toxin; culture food, *not* stool
	Gastroenteritis occurs in 1–6 hours after eating
	Self-limited
Vibrio cholerae	Gram-negative comma-shaped rod
	Enterotoxin stimulates adenylate cyclase in small bowel
	Contracted from drinking contaminated water or eating contaminated seafood, especially crustacea
	Treatment: fluid replacement; glucose and sodium required in oral supplements (cotransport system for reabsorption); doxycycline or fluoroquinolone
Yersinia enterocolitica	Gram-negative coccobacillus with bipolar staining
	Enterocolitis in children; mesenteric lymphadenitis (granulomatous microabscesses) that simulates acute appendicitis
	Association with HLA-B27 positive seronegative spondyloarthropathy
	Treatment: TMP-SMX
Protozoa	
Balantidium coli	Protozoan (ciliate); largest protozoan Transmitted by ingestion of cysts in food or water
	Produces colonic ulcers with bloody diarrhea
	Treatment: tetracycline
Cryptosporidium parvum	Protozoan (sporozoa)
	Transmitted by ingestion of oocysts in food or water
	• Responsible for outbreaks of diarrhea in water supply (e.g., Milwaukee, WI) • Responsible for childhood diarrhea in day care centers
	Most common cause of diarrhea in AIDS
	Diagnosis: stool antigen test (sensitivity/specificity 98%); oocysts partially acid-fast
	Treatment: if immunocompromised, nitazoxanide (less responsive to drug if immunodeficient)
Cyclospora, Microsporidia, Isospora belli	Protozoa (sporozoa) Fecal-oral transmission
	All are common pathogens in AIDS diarrhea
	Cyclospora can contaminate raspberries
	Microsporidia spores are *not* partially acid-fast
	Cyclospora oocysts partially acid-fast
	Isospora oocysts partially acid-fast
	Treatment: *Cyclospora*—TMP-SMX double strength; *Microsporidia*—albendazole; *Isospora*—TMP-SMX double strength

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▼Table 14–6.0B Pathogens and Diarrhea *(continued)*

Pathogen	Discussion
Protozoa ***(continued)***	
Entamoeba histolytica	Protozoa (amoeba)
	Transmitted by ingestion of cysts in food and water
	Cysts are nonmotile and are present in formed stool; trophozoites are motile and are present in diarrhea
	Produces dysentery (bloody diarrhea); cysts exist in the cecum and become trophozoites in the cecum; trophozoites release powerful histolytic agents that produce flask-shaped ulcers; trophozoites can penetrate portal vein tributaries and drain to the liver to produce a liver abscess ("anchovy paste" abscess); trophozoites can penetrate hepatic vein tributaries and produce systemic disease
	Trophozoites characteristically phagocytose red blood cells
	Diagnosis: stool antigen test (sensitivity/specificity 100%)
	Treatment: metronidazole (diarrhea/dysentery; extraintestinal disease); paromomycin (passage of asymptomatic cyst carrier)
Giardia lamblia	Protozoa (flagellate) Most common protozoal cause of diarrhea in U.S.
	• Transmitted by fecal-oral route through ingestion of cysts in food and water • Reservoirs for *G. lamblia* that contaminate water supplies include beaver, voles, and muskrats
	Common in day care centers, mental hospitals, among hikers, water supplies (chlorination does *not* kill the cysts), men who have sex with men (anal-oral contact), IgA deficiency, common variable immunodeficiency
	Produces acute and chronic diarrhea with malabsorption (cysts in formed stool; trophozoites in loose stools)
	Diagnosis: stool antigen test (sensitivity/specificity 100%)
	Treatment: tinidazole or nitazoxanide
Helminths	
Anisakis simplex	Intestinal nematode
	Transmission: eating raw fish dishes (i.e., sushi, sashimi); eating pickled herring
	Larvae penetrate gastric and intestinal mucosa
	Produce cramping abdominal pain; epigastric distress with nausea, vomiting, and diarrhea within a few hours after eating
	Diagnosis: endoscopy; IgE antibody test
	Treatment: removal by endoscope or surgery
Enterobius vermicularis	Intestinal nematode; most common helminth in the U.S.
	Fecal-oral transmission by ingestion of eggs, which are the infective form
	Eggs deposited in anus by adult worms cause pruritus ani
	Other infections: urethritis in girls; acute appendicitis
	No eosinophilia because adult worms are *not* invasive
	Treatment: albendazole or mebendazole

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▼Table 14–6.0B Pathogens and Diarrhea *(continued)*

Pathogen	Discussion
Helminths ***(continued)***	
Trichuris trichiura	Intestinal nematode (whipworm)
	Fecal-oral transmission by ingestion of eggs, which are the infective form
	Produces diarrhea; can produce rectal prolapse in children
	Diagnosis: stool for ova and parasites; eosinophilia
	Treatment: albendazole
Ascaris lumbricoides	Largest intestinal nematode
	Fecal-oral transmission by ingestion of eggs, which are the infective form
	Larval phase through lungs: cough, pneumonitis, eosinophilia (invasion of tissue)
	Bowel obstruction in adult phase; no eosinophilia (no invasion of tissue)
	Treatment: albendazole and mebendazole
Necator americanus	Intestinal nematode (hookworm)
	Transmission by direct penetration or autoinfection; filariform larvae is infected form, adults attach to villi, resulting in blood loss; thus iron deficiency
	Treatment: albendazole or mebendazole
Strongyloides stercoralis	Intestinal nematode
	Transmission: filariform larvae in soil penetrate the feet → larval phase through the lungs → swallowed and molt into adults that enter the intestinal mucosa and lay eggs → eggs hatch into rhabditiform larvae, which enter the intestinal lumen and are passed in the stool → develop into filariform larvae (infective form) in the soil
	Autoinfection occurs if filariform larvae penetrate intestinal mucosa and migrate to the lungs
	In immunocompromised patients, reinfection occurs with dissemination throughout the body
	Produces abdominal pain and diarrhea
	Treatment: ivermectin
Diphyllobothrium latum	Intestinal cestode (tapeworm)
	Transmission by ingestion of sparganum larvae; found in trout of the Great Lakes region
	Diarrhea with or without vitamin B12 deficiency; preferential uptake of vitamin B12 by the worm
	Diagnosis: eggs in the stool
	Treatment: praziquantel
Hymenolepis nana	Intestinal cestode
	Dwarf tapeworm, commonly found parasite in house mice worldwide
	Fecal-oral transmission; eggs hatch in the small intestine → oncosphere burrows into villi and develops into cysticercoid larva → larva breaks out into lumen to become adult worm
	Asymptomatic or may have abdominal pain or diarrhea
	Treatment: praziquantel
Hymenolepis dimunta	Intestinal cestode
	Common parasite in rat droppings, which contain eggs; flour beetles or moths ingest the eggs, and cysticeroid larvae develop in these arthropods; humans ingest the infected beetles/moths in the flour and larvae develop into adult worms in the human intestine
	Asymptomatic or have abdominal pain or diarrhea
	Treatment: praziquantel

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